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BEFORE THE BOARD OF PATENT APPEALS AND INTERFERENCES

Application Number: 09/820,099
Filing Date: March 27, 2001
Appellant(s): VAN DE WINKEL, JAN G.J.

Ms. Jeanne M. DiGiorgio
For Appellant

EXAMINER'S ANSWER

This is in response to the appeal brief filed 7/16/04.

(1) Real Party in Interest

A statement identifying the real party in interest is contained in the brief.

(2) Related Appeals and Interferences

The brief contains a statement that there are no appeals or interferences known to Appellant or Appellant's legal representative.

(3) Status of Claims

The statement of the status of the claims contained in the brief is correct.

(4) Status of Amendments After Final

The appellant's statement of the status of amendments after final rejection contained in the brief is correct.

(5) Summary of Invention

The summary of invention contained in the brief is correct.

(6) Issues

The appellant's statement of the issues in the brief is correct.

(7) Grouping of Claims

The rejection of claims 1 and 6-12 stand or fall together because appellant's brief does not include a statement that this grouping of claims does not stand or fall together and reasons in support thereof. See 37 CFR 1.192(c)(7)

(8) Claims Appealed

The copy of the appealed claims contained in the Appendix to the brief is correct.

(9) Prior Art of Record

Shen et al., WO 98/23646

Monteiro et al., J. Exp. Med. 171:597-613, 1990

(10) Grounds of Rejection

The following ground(s) of rejection are applicable to the appealed claims:

Claims 1, 6-12 are rejected under 35 U.S.C. 103(a) as being unpatentable over Shen et al (WO 98/23646, published 6/98, IDS #4) as evidenced by Monteiro et al (J. Exp. Med 171:597-613, 1990) and the specification.

The claims recite a method for eliminating a target cell or antigen from the circulatory system of a subject comprising administering a complex comprising monomeric IgA or a portion that binds to FcαRI linked to a second portion that binds a cancer target cell or antigen or bacteria, fungus, or virus, further claimed is that the method comprises administration of GM-CSF, wherein administration is by intravenous.

Shen et al teach binding agents specific for the FcαR and the binding agents triggers an Fc mediated effector cell activity such as phagocytosis (see page 1). Shen et al also teach bifunctional binding agents comprising an agent that binds FcαRI and a bacteria (see page 22) or cancer cell or antigen (see page 19-20) thereof, further is a method for eliminating cells or antigen in a subject by administration of the bispecific agent to a subject (see page 28-29) and the method further comprises adding GM-CSF which enhances the number or activity of Fcα receptors (see page 28) and the method comprises administration intravenous (see page 29, line 35) and the binding agents bind the FcαR with the same affinity as a type of IgA which can be monomeric IgA (see page 6). As evidenced by Monteiro et al (and the specification at page 1, lines 6-8) there is only a single class of IgA Fc receptor, Fcα RI, therefore since the agent binds to

Fcα RI, it would be obvious that the agent would bind to Fcα RI expressed on Kupffer cells and adding cytokine would increase the expression of the Fcα RI on Kupffer cells. Shen et al does not specifically teach that the binding agent can be monomeric IgA.

It would have been prima facie obvious to one of ordinary skill in the art at the time the claimed invention was made to have used the complex comprising monomeric IgA linked to a second antibody (a bispecific agent) for the elimination of a target cell or antigen.

One of ordinary skill in the art would have been motivated to and had a reasonable expectation of success to have used the complex comprising monomeric IgA linked to a second antibody (a bispecific agent or multispecific) for the elimination of a target cell or antigen because Shen et al teach FcαRs are capable of interacting with IgA in the form of monomers and binding induces phagocytosis (see page 3, lines 28-30) and Shen et al teach that the binding agent binds with the same affinity as monomeric IgA and that the binding agent does not inhibit the binding of IgA (see page 5-6). Thus, it would have been obvious to have the binding agent be monomeric IgA linked to a second antibody because monomeric IgA would bind with the same affinity as a type of IgA and it would bind to the IgA site and perform phagocytosis.

Therefore, the invention as a whole was prima facie obvious to one of ordinary skill in the art at the time the invention was made, as evidenced by the references.

Response to Arguments

The Brief of page 4-5 states that the Examiner's rejection is based on the erroneous assumption that it would have been obvious to have substituted monomeric IgA as the binding agent for FcαR in the bispecific molecule taught by Shen et al and in contrast to the binding agents for FcαR taught by Shen et al, monomeric IgA naturally functions as a bispecific molecule that binds to FcαR and a target antigen. In addition, the Brief states "as discussed by Shen et al., monomeric IgA binds to FcαR via its constant region and to a target antigen via its variable region" (see page 4 of Brief).

In response to these arguments, while monomeric IgA may be bispecific, it does not rule out using it as the binding agent in Shen et al because Shen teaches binding agents that are a combination of binding agents such as a first binding agent having binding for FcαR and a target antigen and a second binding agent for an antigen binding region to a different epitope of the same target antigen (see page 28 lines 10-15) of which multispecific agents are contemplated having three different binding regions (see page 1, lines 30-37, page 6, lines 6-21) . With regard to the statement that monomeric IgA binds to FcαR via its constant region and to a target antigen via its variable region in Shen et al, again as stated in the final rejection Office Action, it is unclear where Shen teaches such. Shen teaches FcαRs are capable of interacting with monomeric IgA and inducing phagocytosis (see page 3, lines 28-30). Thus it was known that monomeric IgA would bind and induce effector functions.

The Brief on page 5 further states that Shen teaches away from using monomeric IgA. The Brief states that Shen et al teach targeting antigens is made more effective by using a binding agent that does not compete with natural ligand for binding

to FcαR and binds a site distinct from the IgA binding site. In response to this Shen does not state that monomeric IgA could not be used. The passage states that a preferred embodiment is that the binding agent is not inhibited by binding of IgA to the receptor, but this is in the context of an antibody as the binding agent. The detailed description of the invention of Shen states (page 3, lines 2-3) that "The invention pertains to a binding agent having at least one antigen binding region specific for an IgA receptor" and as stated in Shan et al and in the Brief by Appellants (page 4-5) monomeric IgA has this function. Therefore it is obvious to use monomeric IgA as the binding agent.

The Brief on page 5 further states that there would have been motivation to substitute dimeric not monomeric IgA because the role of monomeric IgA was poorly understood. In response to this argument, Shen specifically teach that monomeric IgA was known to interact with receptors and cause phagocytosis, therefore, it would have been prima facie obvious to one of ordinary skill in the art at the time the claimed invention was made to have used the a complex comprising monomeric IgA linked to a second antibody (a bispecific agent or multispecific) for the elimination of a target cell or antigen.

For the above reasons, it is believed that the rejections should be sustained.

Respectfully submitted,

LARRY R. HELMS, PH.D
PRIMARY EXAMINER

Larry R. Helms
August 12, 2004

Conferees

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